

THE PRESENCE OF A DIAGONAL EAR-LOBE CREASE AS AN INDICATOR  
OF CORONARY ARTERY DISEASE

by  
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This thesis has been read by each member of the following supervisory committee and by majority vote has been found to be satisfactory.

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FINAL READING APPROVAL

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I have read the thesis of Grace Blodgett in its final form and have found that (1) its format, citations, and bibliographic style are consistent and acceptable; (2) its illustrative materials including figures, tables, and charts are in place; and (3) the final manuscript is satisfactory to the Supervisory Committee and is ready for submission to the Graduate School.



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## ABSTRACT

The purpose of this study was to determine whether a significant difference existed in the occurrence of an ear-lobe crease (ELC) either bilaterally or unilaterally, between those individuals who had sustained a myocardial infarction (MI) and those who had not. The rationale for the study was based on the assumption that the ELC would serve as an indicator for those individuals who are likely to experience coronary artery disease (CAD).

The hypotheses pursued were that a) a positive correlation existed between the presence of an ELC and CAD, b) a positive correlation existed between the presence of the ELC and some of the risk factors for CAD, i.e., sex, heredity, cigarette smoking, hypertension, elevated serum glucose, obesity, aggressive personality (stress) and elevated serum cholesterol; c) Males demonstrated a greater prevalence for the ELC than females, and d) Caucasian, American culture increases the incidence of the ELC as opposed to any other culture.

Seventy-seven individuals who had sustained an MI and 77 individuals who had not were observed for the presence or absence of an ELC. Among these two groups, individuals with risk factors for CAD (including age, sex, heredity, obesity,

cigarette smoking hypertension, elevated serum glucose, aggressive personality and elevated cholesterol) were observed for the presence or absence of an ELC.

The findings were tested using chi-square, with Phi, Cramer's and contingency correlation coefficients used to demonstrate strength of any demonstrated correlation. The results indicated that the presence of the ELC demonstrates a statistically significant (positive) correlation with CAD, age, hypertension and elevated serum cholesterol levels. There was no correlation with sex, heredity, elevated serum glucose levels, stress, obesity, and cigarette smoking.

These findings indicated that it is possible to predict that people who have an ear-lobe crease are also likely to experience CAD. The detection of an ELC (as an indicator of possible CAD) is a simple, inexpensive, noninvasive sign to be used as a motivator in the education process to modify coronary risk factors.

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## CHAPTER I

### INTRODUCTION

Coronary artery disease (CAD) and resultant myocardial infarctions (MIs) are a leading cause of death and disablement among men and women in the U.S.A. today, although women to a lesser degree than men. Myocardial infarctions (MIs) were directly attributed to the deaths of 646,703 persons during 1976, representing 64.8% of all cardiovascular deaths. Coronary artery disease affects 4,190,000 Americans annually, of whom approximately 1,000,000 will suffer a myocardial infarction (MI) (American Heart Association, 1979).

Numerous long-term studies have demonstrated a correlation between the presence of what have been identified as risk factors and the increased incidence of coronary artery disease (Epstein, Ostrander, Johnson, Payne, Hayner, Keller & Francis, 1965; The Framingham Study, 1974). Risk factors are factors which contribute to the atherogenic process and therefore, MIs, and may be inherited or acquired, nonmodifiable or modifiable. At least 185 distinct, but related, factors have been suggested to have either positive or inverse associations with coronary artery disease (CAD) (Hopkins & Williams, in press). From these

185 factors, the following 13 have been referred to as major risk factors which are strongly predictive of the possibility of future CAD: age, sex, family history, glucose intolerance, hypertension, obesity, elevated cholesterol, sedentary living, aggressive and competitive personality, abnormal lipoprotein metabolism, abnormal platelet aggregation and genetic influences.

The study by the U.S. Department of Health (The Framingham Study, 1974) involving the people of Framingham, Massachusetts demonstrated that the predisposition of an individual towards CAD increases greatly with each additional risk factor and the effects are cumulative. Those risk factors which are most commonly involved on a cumulative basis are hypertension, hypercholesteremia, diabetes mellitus, a positive family history and male sex. Williams (1980) reported that approximately 50% of the time, these characteristics are largely determined by inherent genetic makeup. He further states that some of these risk factors and the susceptibility of an individual to CAD are thought to "be determined by an interaction between environmental influences modulated by inherent genetic makeup" (Williams, 1980, p.333). The exact genetic mechanisms involved, which cause one individual to respond to specific environmental factors and develop CAD and another individual to show no response are, as yet, unknown. Williams, using the geneological information available in Utah, has four years of accumulated

data on over 1,000,000 people in Utah, extending back four to six generations. Of the 140,752 deaths reported, 37,942 were coronary heart disease deaths. Over half of the deaths with CHD had a positive family history; diabetes and hypertension being present in 50% of the female deaths and hypercholesterolemia found in 25% of both male and female deaths. Williams' Utah project is attempting to uncover more information on this promising unknown genetic factor.

Over the past decade there has been a concerted effort by health care professionals to educate the public and bring to their awareness evidence that these risk factors do exist and indeed play a great role in the development of CAD. This educational process has involved an emphasis on the fact that the majority of these identified risk factors can be modified and reduce the effect that they have on the atherogenic process. It is suggested that a slowing or reduction of this atherogenic process lessens the possibility of an MI.

Members of the nursing profession play a vital role in this education process, as they provide much of this education to the public both directly and indirectly, formally and informally. Nurses perform client assessments, evaluate, make nursing diagnoses, plan and refer, in addition to the education process. The nurse often facilitates an entrance for the client to the health care system to receive appropriate assistance, regardless of the discipline involved. Considered by some to be a

coordinator of these many disciplines, the nurse is in the most advantageous of positions to refer the client to the physician or to a supportive discipline.

The speculation that an association exists between CAD and an ear-lobe crease has been discussed since the late 1960s. Frank (1973) first offered a description of the ear-lobe crease and identified a possible connection between its presence and CAD: "A presumptive relationship which warrants testing" (Frank, 1973, p. 327). At the time of his writing, Frank admitted that the observations were not controlled, but nevertheless, he found the results striking and strongly suggestive that there was some correlation between CAD and the presence of an ear-lobe crease (ELC) (For illustration, see Appendix D).

The implications for nursing, if these positive correlations can be demonstrated, are readily apparent. The presence of a diagonal ELC, either unilateral or bilateral, would indicate that these individuals might be at high risk for CAD prior to the morbid occurrence of an MI. The emphasis would then be a preventative, educational process to assist the individual to change his/her lifestyle by treating and/or reducing those risk factors which can be modified.

#### Problem Statement

The problem investigated in this project was:

Is there a positive relationship between coronary artery disease and the presence of a unilateral or bilateral ear-lobe crease in individuals who have experienced a myocardial infarction?

### Purpose

The purpose of this study was to determine whether a significant difference existed in the occurrence of an ear-lobe crease, either bilaterally or unilaterally, between those individuals who have sustained a myocardial infarction and those who have not. The results of this study suggest that the ELC could be used as a screening tool to identify those individuals who might be at high risk for coronary artery disease .

The significance of this determination for nursing practice and public education lies in the fact that the ELC would be an inexpensive, easily performed, noninvasive procedure. It would serve as a method by which the health professional might reach those individuals with one or more of the risk factors for CAD. A preventative approach to the education of the high risk patient, instead of a postmyocardial infarction approach would be beneficial to the individual and to society as a whole.

### Relationship to Nursing Conceptual Framework

Since cardiovascular disease accounts for 56% of adult deaths in America, it presents one of the greatest challenges



that the health profession is faced with today. One aspect of cardiovascular disease is atherosclerosis and although great advances have been made over the past two decades, a tremendous amount of unknowns persist. The actual cause of the atherosclerotic process has yet to be determined. Although there are numerous contributing factors to atherosclerosis and CAD, the actual key to the disease process, and thereby, the actual "cure" has yet to be identified.

Current research suggests that there may be unknown genetic and/or anthropological factors involved in the atherosclerotic process (Hopkins & Williams, in press) and subsequent CAD. It is suggested that these same factors are also involved in the formation of the ear-lobe crease and that the genetic influence exerted upon an individual to predispose to CAD has a strong correlation with the influence exerted towards the presence of an ELC.

There are certain interrelated concepts fundamental to the suggestion that there is a positive correlation between the ELC and CAD (see Figure 1).

1. Coronary artery disease and the ELC may share a genetically transmitted factor, the exact D.N.A. coding or mechanism of which is unknown.
2. Hypertension, diabetes mellitus, obesity and sex are risk factors for CAD of which approximately 50% are determined by genetic influences.

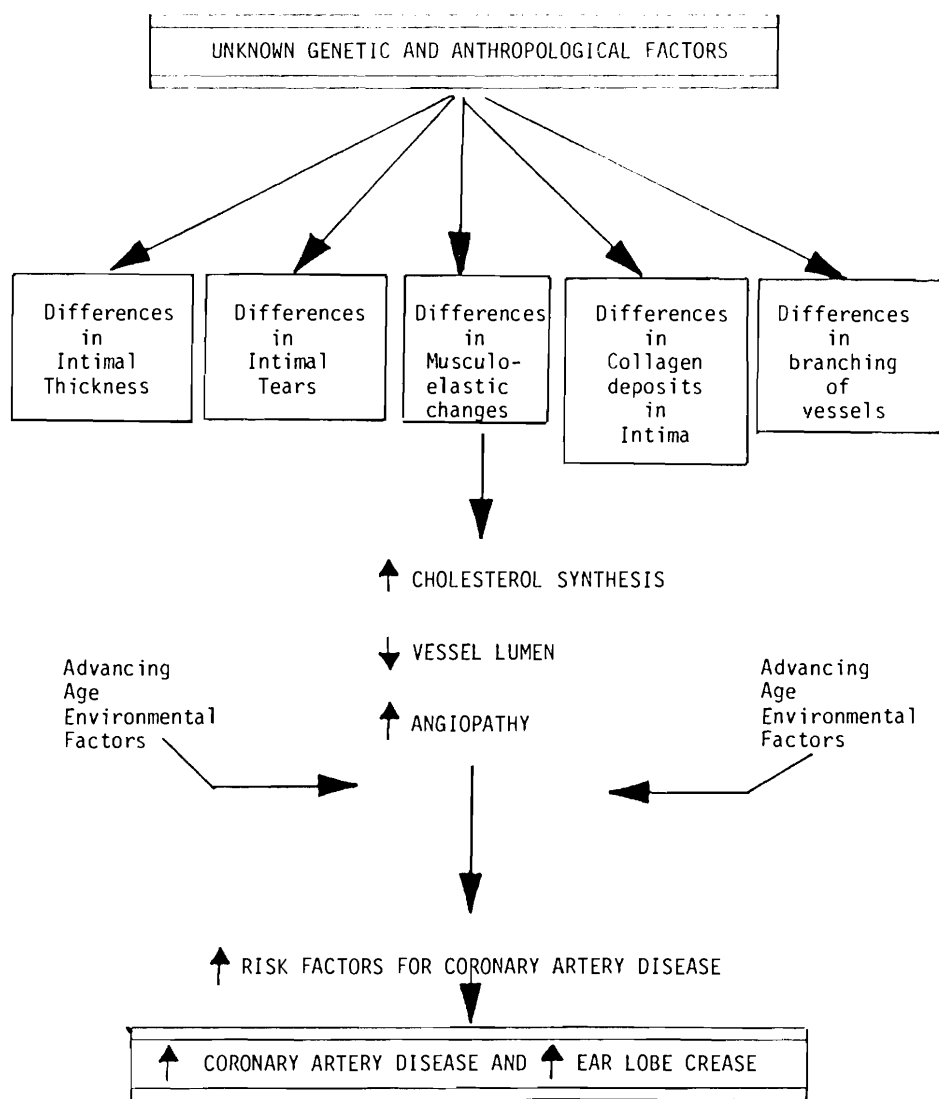


Figure 1. Conceptual model.

3. Hypertension, diabetes mellitus and obesity have each been identified with the presence of the ELC.
4. Anthropological factors may influence coronary artery branching and vascularization, collagen deposits on the intima, musculoelastic layers within the vessel, and the incidence of the ELC.
5. Collagen vascular diseases may promote changes in the connective tissue of the arterial walls, resulting in sclerosis and narrowing of the arterial vessels.
6. The greater the amount of sclerosis and occlusion within the coronary vessels, the greater the chance for the presence of bilateral ELCs.
7. The ELC and CAD share age as a common denominator.
8. The external diameter, the medial thickness and the internal thickness of the arterial vessels increase progressively with age; males at a greater rate than females.
9. A diminished blood supply to the highly vascular ear lobe may contribute to the elastic fiber tears which are manifest as the ear-lobe crease.
10. The ear-lobe crease may be the result of a pathological process of small vessels similar to that found in hypertension, diabetic retinopathy, and CAD.

The interrelated concepts depicted previously demonstrate that CAD and the ELC share a number of common factors, but the

question and initial cause remains unanswered. It was postulated that this single cause of CAD and the ELC is one or more of the following:

1. Genetically and/or anthropologically determined;
2. A collagen-vascular disease, e.g., changes in elastin, sclerosis of arterial walls, thickness of internal and medial layers and elastic fiber tears.
3. A response to injury.

However, as shown in Figure 1, the common factors seen in CAD and the ELC are the primary nursing concern. Identification of the cause of the correlation is less important than that the correlation exists. With the establishment of a positive correlation, the ELC has been demonstrated to be a useful, readily visible indicator of individuals at high risk for CAD.

## CHAPTER II

### REVIEW OF THE LITERATURE

#### Demonstrating the Relationship of Coronary Artery Disease to the Ear-Lobe Crease

Early studies indicated that some relationship exists between Coronary Artery Disease and the ear-lobe crease but the significance of this correlation varies from study to study.

The initial observations of the ELC and CAD performed by Frank (1973) were restricted to a limited number of patients, a total of only 20. He observed 20 individuals with the ELC and all but one had one or more risk factors present. Five individuals had one risk factor, seven individuals two risk factors, one individual three risk factors, three individuals four risk factors, and one individual had five or six risk factors. The risk factors observed were: elevated cholesterol, triglycerides and blood sugar, hypertension, cigarette smoking and a positive family history. This observation by Frank has led to much speculation and a minimal amount of research. The results of the research thus far have demonstrated some aspects of support for his observations and some aspects which do not support his observation.

support his observation.

Since Frank first identified the ELC as a possible predictor of CAD, suggestions have been put forward by some investigators that the ELC has much significance in other disease entities, such as diabetes mellitus and hypertension. It is of interest to note that all the diseases included, or also having some connection with the ELC, are all included in the list of major risk factors for CAD.

It is essential to emphasize that the ELC is a biological variation from the norm which is not present at birth, but which develops with advancing years. The ELC being studied bears no connection with the congenital slit-like indentations of the earlobe (known as Kerbenohr indentations) characteristically found in children with Beckwith's syndrome (Wideman, 1979) (neonatal hypoglycemia occurring in infants with visceromegaly, microcephaly, facial nevus and other minor defects).

One of the earliest studies performed for the ELC and CAD was done at Mount Sinai Hospital, New York City, from July 1970 through June 1973 (Lichstein, Chadda, Naik & Gupta, 1974). Five hundred and thirty-one post-MI patients were observed in a coronary care unit against a control group of 305 non-MI patients. Questionnaires were completed at the time of admission determining the presence or absence of an ELC, hypertension, diabetes, and smoking habits. The ELC was evaluated as being present either unilaterally or bilaterally.

Diabetes mellitus was evaluated as present if the patient had a history of taking insulin or oral hypoglycemic agents or a fasting glucose consistently abnormal (greater than 120 mgms per 100 ml). Hypertension was evaluated as present if the patient had a history of more than one blood pressure of 150/100 mmHg or taking antihypertensive medications.

The study group, when observed, was noted to have 251 individuals with an ELC which represented 47% of the 531 observed. The control group had 92 out of 305, representing 30%. The study group had a greater number of individuals with an ELC than the control group in all age categories, but the difference was most apparent in the sixth to ninth decades. The authors claimed that 47% to 30% was highly significant ( $p < 0.001$ ). The authors chose not to have the same control group numbers as study group numbers which makes the study results less reliable.

In the study group with CAD, diabetes mellitus was present in 16% of the individuals in the experimental group and present in 11% of the individuals in the control group. Hypertension was present in 16% of the experimental group and present in 12% of the control group. Cigarette smoking was present in 42% of the individuals in the study group and present in 39% of the individuals in the control group.

When these same three variables were observed in relationship to the presence of the ELC, the findings were not

particularly indicative of any correlation. In the study group, diabetes was present in 18% with the ELC, and present in 13% without the ELC. The control group demonstrated similar findings with diabetes present in 15% with the ELC and nine percent without the ELC. Hypertension in the study group was found to be present approximately 28% of the time with or without the ELC being present. The control group individuals demonstrated hypertension present in 17% of those with the ELC, and 10% of those without. The incidence of smoking in the study group patients with an ELC was significantly higher, 68% to 56%. However, in the control group no significant difference was found in individuals with or without the ELC. From the figures obtained, Lichstein et al. stated that the ELC was not simply a result of diabetes, hypertension, or cigarette smoking, but that its presence in CAD "seems clear" (Lichstein et al., 1974, p.615). The authors are to be commended on their relatively large study group so early following the initial suggestion of a correlation, however, an equal number of control group individuals would have increased reliability.

Anderson, Mathiesen, Calberg and Christiansen (1975) observed all the patients admitted to the medical/surgical departments in a Danish hospital over a period of two months; 203 women and 320 men. As in the study by Lichstein et al. these subjects were observed for MI, ELC, hypertension, diabetes mellitus and smoking habits. Anderson et al. did not find the ELC to be



present before the age of 30 and only in three patients between the ages of 30 and 49 years. After that age an "increasing frequency" was found, although the authors did not elaborate further. They found that the prevalence of the ELC was greater in the group of 50 years old with MI and CAD than the control group. A higher correlation between CAD and the ELC was found, more so than diabetes, hypertension and cigarette smoking and the ELC as a risk indicator. Their findings were not detailed, nor was the actual study well outlined in the abstract. It would appear that their findings were similar to those obtained by Lichstein et al. (1974).

Sternlieb, Gau, Davis, Rutherford and Frye (1974) performed a study at the Mayo Clinic in which 144 patients were observed for the presence or absence of an ELC. Of the 144 observed, 133 had an ELC present. Of the 144 observed, 120 had CAD, findings which Sternlieb et al. (1974) found to be "significant." The article did not mention a control group, which is unfortunate, for the figures obtained, although significant, had no comparison or referent group to add weight to the findings. A lack of observation of age also demonstrated a weakness in the study.

#### Current Studies Relating CAD to ELC

A study of apparent depth was performed by Kaukola, Manninen, Valle and Halonen in Finland (1979). Two hundred and eighty-six patients were randomly selected who had undergone

selective coronary angiography because of typical signs and symptoms of coronary heart disease (CHD) or atypical chest pain. Of the 286 patients observed, 200 had at least 50% occlusion in one of the main epicardial arteries (CHD patients) and the remaining 86 patients demonstrated no significant stenosis (non-CHD patients). Both the CHD group and the non-CHD group age ranges fell between 26-66 years, with the mean for the CHD group being 48 and for the non-CHD group, the mean was 50. However, the percentage of males in the CHD group was markedly higher than the non-CHD group, 63% compared with 13%.

Kaukola et al. (1979) observed both groups for the presence or absence of an ELC. One hundred and forty-four of the 200 CHD patients had an ELC (72%), while only 18 of the non-CHD patients had an ELC (21%). As demonstrated by Lichstein et al. (1974) and Anderson et al. (1975), the prevalence of the ELC increased with age. Kaukola et al. found the ELC in almost 90% of the CHD patients with triple vessel disease in the 50-59 years old group. It is of interest to note that six of the non-CHD patients (those without stenosis) had previously sustained an MI, five of which had an ELC present.

The Kaukola study observed the patients for increased serum cholesterol and triglycerides, low HDL-cholesterol, hypertension, cigarette smoking, diabetes and obesity. Those factors were more commonly seen among the CHD patients, as was to be expected, but multivariate analysis showed that none were

associated with the ELC.

Coronary angiography and observation of the ELC were considered to be reliable, objective measures, and no other factor affected the selection of patients. In view of the fact that the patients were divided into the two groups, CHD and non-CHD, according to significant stenosis or negligible stenosis, the results were highly significant. As stated in the study, the authors were restricted to selective cases, due to the invasive nature of angiography which explained the smaller group. It is questionable whether the smaller control group and non-age matched control group affected these findings, which as previously stated, were highly significant.

The subjects selected in the Kaukola study were relatively young (the eldest being 66 years), and although the greatest incidence of the ELC occurred between the years 50 and 59, this does not fully support the findings of the previous studies of the ELC occurring with advancing age. A study which was performed in Oregon also found that age alone did not account for the ELC, and that the ELC "seemed to be best correlated with CHD" (Doering, Ruhsenberger & Phillips, 1977, p.183). Over a ten week period, a study group of 50 with a history of CAD and a control group of 38 without CAD were observed for the ELC. The study group included 30 males and 20 females, the control group 21 males and 17 females. As with the previous studies, the subjects were also observed for the ELC in relationship to

hypertension, cigarette smoking, diabetes, and serum cholesterol but the study also included obesity.

The findings of this study by Doering et al. (1977) demonstrated no significant differences between the two groups except in the presence of the ELC. Of the 50 individuals in the study group, 44 had the ELC (88%). Of the 38 individuals in the control group, 14 had the ELC (36.8%). The authors then structured the study and control groups to include only those who were male, hypertensive, obese, diabetic, cigarette smokers and had the ELC. Eighteen pairs were matched "blindly" by one of the authors according to age, sex and the above-mentioned factors. This time the groups differed only with regards to weight and the ELC. The study group had 17 out of 18 with the ELC (94.4%) and the control group, 8 out of 18 (44.4%). The authors acknowledged at that time that the incidence of the ELC increases with advancing age, but that age alone does not account for the ELC and CHD. Doering et al. suggested that the ELC and CHD are both functions of changes occurring in the vascular system, and the ear lobe may show these vascular changes by starting to crease and fold.

In 1976, Kaukola (1978) performed an extensive two-part study of the ELC association with CAD in Helsinki. The first part of the study involved the observation of 219 acute MI patients and an age and sex-matched control group of 290. Of the study group, 165 were men aged 32-65 years and 54 were women

aged 43-65; individuals over age 65 were excluded from the study. As can be seen, both the male and female controls were well matched with the study group.

Both groups were observed for the ELC, family history of CAD, diabetes, birthplace, previous angina pectoris and MI, hypertension, other cardiovascular disease, diabetes, smoking habits, physical activity at work and during leisure, and lastly, previous drug treatment. Height and weight were recorded. Blood glucose and serum cholesterol were determined two months after the acute attack to avoid MI changes to these levels.

The ELC was found in 151 of the 219 MI patients (69%) and, surprisingly, the same percentage (37 patients) was found in both the men and the women. Kaukola did not find that the increased incidence of the ELC was statistically significant. The youngest man was 32 years and the youngest woman, 43 years, and they were also the youngest MI patients of either sex.

In the control group, the ELC was present in 71 of the 290 subjects (24%) occurring in 66 of the 236 males (28%) and only five of the females (9%). The youngest man with the ELC was 34 years and the youngest woman 57, contrasting with 34 and 43 in the study group. Kaukola did find that the bilateral ELC was more common in men aged 50 and over.

Table 1 demonstrates the risk factor correlation with the ELC in the study group, and the findings were not particularly

significant. Table 2 depicts the risk factor correlation with the ELC in the control group and the findings also were not particularly significant. Additionally, differences in the serum cholesterol, serum triglycerides, body weight and level of activity, in both the study and control groups were not significantly associated with the ELC.

As depicted in Tables 1 and 2, the overall results of the first part of the Kaukola study demonstrated no correlation for the ELC and the risk factors for CAD as single entities. However, the ELC was present 69% of the time in the MI patients and 24% of the time in the control group.

As indicated previously, when the experimental group was limited to individuals who had CAD, as determined by angiography (200 patients), the ELC was present in 144 (72%) of the individuals. The ELC was found to be present in only 21% of the non-CAD individuals. Table 3 demonstrates that the presence of the ELC increased with the amount of vessel disease, the two and three vessel diseases being almost identical. Kaukola (1978) indicated that the ELC increased with age, although specific details were not available.

Table 4 depicts the incidence of the ELC (in relationship to the risk factors) for the 200 individuals who underwent angiography and demonstrated vessel disease, none demonstrating

Table 1  
Study Subjects (Patients with Coronary Heart Disease)  
(n = 286)

Risk Factor	Hypertension	Smoking Habits	Diabetes	Family Hx CAD
ELC	34%	23%	22%	59%
No ELC	38%	15%	21%	61%

Note. Adapted from Kaukola, 1978.

Table 2  
Control Subjects (No Coronary Heart Disease)  
(n = 286)

Risk Factor	Hypertension	Smoking Habits	Diabetes	Family Hx CAD
ELC	15.5%	22.5%	7.0%	30.0%
No ELC	20.0%	32.0%	4.0%	33.0%

Note. Adapted from Kaukola, 1978.

Table 3  
Degree of Coronary Atherosclerosis (As Determined by  
Angiography) and the Presence of the ELC  
(n = 200)

Amount of Atherosclerosis	1 vessel	2 vessels	3 vessels
ELC	52%	79%	79%
No ELC	48%	21%	21%

Note. Adapted from Kaukola, 1978.

Table 4  
CAD Patients with Demonstrated Vessel Disease  
(n = 200)

Risk Factor	Hypertension	Smoking Habits	Diabetes	Family Hx CAD
ELC	34%	42%	21%	76%
No ELC	23%	41%	23%	75%

Note. Adapted from Kaukola, 1978.



particular significance. Again, as in the patients with all forms of coronary heart disease (CHD), the serum cholesterol, serum triglycerides, serum lipoproteins, and body weight differences were not significant.

It is interesting to note that of the 33 patients with CHD who were not included in the CAD category, 24 experienced intermittent claudication. When all 286 CHD patients were interviewed, 29% of those with the ELC and 7% without the ELC responded that in their history previous to angiography they had also experienced intermittent claudication.

During this study, Kaukola also performed postmortems to identify any histological changes in the lobular portions of the ear. To explain the ELC formation, ten post-MI patients with ELC were compared with ten control patients; none of the MI patients were without the crease. Distended capillaries, some nonspecific inflammatory changes, and collagen degeneration were found. Both groups were the same except that one person who had had an MI demonstrated significant sclerosis of the small arteries of the lobule.

The Kaukola study was performed in great depth with a definite correlation demonstrated between CAD and the ELC, but no correlation between the risk factors for CAD and the ELC. It is unfortunate that with such a detailed and obviously well performed study, the control group could not equal the study group although statistically and ethically the low number

is acceptable.

A similar study to Kaukola's (1978) (in the postmortem examination only) was performed by Lichstein, Chapman, Gupta, Chadda, Smith, Schwartz and Naik (1976). They examined the relationship of the ELC and CAD still further, comparing at postmortem the status of the ELC with the degree of sclerosis and the degree of coronary occlusion.

One hundred and thirteen consecutive patients, aged 40 or older, were examined at postmortem. Immediately prior to the postmortem, the patients were categorized as having the ELC present either unilaterally, bilaterally or absent. The coronary arteries were opened by an examiner unaware of the ELC findings, and evaluated for degree of sclerosis of the arterial wall and patency of the lumen. Of the 113 patients observed, 32 did not have an ELC, 22 had a unilateral ear crease and 59 had bilateral ear-lobe creases. No significant difference was found between sex, risk factors, and the ELC; however, a significant difference ( $p < 0.01$ ) was found between the amount of sclerosis of the coronary arteries in the no ELC group when compared with the bilateral ELC group. The degree of occlusion was greatest in the bilateral ELC group, less in the unilateral ELC group, and lowest in those with an absent ELC.

The findings obtained by Lichstein et al. (1974) and Anderson et al. (1975) when observing diabetes and the ELC did not demonstrate any correlation. The same assumption was held

by Andreson, Christiansen and Jensen (1976) who examined 101 patients, 45 women and 56 men with diabetes mellitus, none of whom were under the age of 30 years. The retinal vessels were examined while the patients were having routine observations and control of their diabetes. Of those examined, 35 had retinal angiopathy and 66 did not. Of the 35 with angiopathy, 51.4% (18) had the ELC, and only 7.6% (5) of those without retinal angiopathy had the ELC. The age within both groups did not differ significantly. The authors felt that, in addition to coronary angiopathy (from previous observations), there is a positive correlation between retinal angiopathy and generalized angiopathy.

#### Relationship of ELC and CAD Risk Factors

Obesity, although not clearly demonstrated as a risk factor for CAD, per se, is usually included because of the influence it exerts on diabetes, hypertension and sedentary living, which are identified as risk factors for CAD. Rhoades, Klein, Yano and Preston (1977) observed 1237 middle aged Japanese men for obesity and the ELC. Upon completion of their study, they stated that there was a positive correlation with obesity, a weak correlation with hypertension, yet no CAD-ELC relationship was demonstrated. It was of interest to note that the incidence of CAD and the ELC are relatively infrequent in Japan in comparison with other countries.

Hypertension has long been identified as a major risk factor for CAD and, as such, has been observed for a positive relationship with the ELC. Frank's (1973) initial observation of 20 individuals with the ELC revealed that 75% were hypertensive, "a significant finding." However, the study number was small and there was no control group with which to compare, so its significance might be questioned.

Petrakis presented an interesting perspective in his writings on the Emperor Hadrian and the ELC. Busts of Hadrian presented in European museums clearly depict an ELC bilaterally, on four different busts. Hadrian had a Type A personality, or "the type of personality one needs to be an emperor" and from historical writings reviewed, Petrakis suggests he had hypertensive and arteriosclerotic heart disease. Hadrian died at age 62, reducing age as the single cause of the ELC. This is only a provocative observation.

Recently, Schoenfeld, Mor, Weinberger, Avidor and Pinkhaus (1980) conducted a study on 421 MI patients, along with a control group of 421 sex, age and ethnic origin matched individuals. The researchers attempted to correlate the ELC with hypertension, diabetes, smoking, hyperlipidemia, and also included hyperuricemia. A highly significant percentage of the MI group had the ELC (77%) in comparison to 40% of the control group. No significance was established between the vessel site of the MI, the ELC, or the sex of the individual. Significant

association was, however, found between some of the risk factors and the ELC as shown in Table 5. Specifically, hypertension, diabetes, diabetic retinopathy, and elevated cholesterol were much more evident in patients with the ELC when compared with those without. No significant association was found between hypertensive retinopathy, hyperuricemia, hypertriglyceridemia, cigarette smoking and the ELC.

The data demonstrated an increased frequency of the ELC in the MI group, regardless of age. The two groups were divided into sections, according to age, 30-50 years, 51-60 years, 61-70 years, 71-80 years and 81-90 years. In each age section, the incidence of the ELC in the MI group was above 70%, whereas in the control group it ranged from 25-45% only, thus not supporting the claim that the ELC is age-related only. It did support a statement by Frank (1973) that "the ELC is a pathological process of small vessels found in CHD, hypertension and diabetic retinopathy" (p 327). Prevalence of the ELC increased with the extent of atherosclerosis. Single vessel disease individuals had 52% with the ELC and double or triple vessel disease individuals, 79% with the ELC.

#### Use of Ear-Lobe Crease as Diagnostic Tool

In an attempt to use the ELC as a diagnostic tool, two studies were performed using the ELC as a tool for screening. The first study, conducted by Sprague (1976) used the ELC as an indicator of operative risk. Sprague observed 222 elective

Table 5  
 Relationship of ELC to Risk Factors in Patients with  
 Recent Myocardial Infarctions  
 (n = 421)

Risk Factors	Hypertension	Diabetes	Diabetic Retinopathy	Increased Cholesterol
ELC	86.8%	84.6%	96.0%	82.4%
No ELC	13.2%	15.4%	4.0%	17.6%

Note. Adapted from Schoenfeld et al., 1976.

surgery patients from the day of admission to discharge for the presence or absence of ELC, clinical CAD, and intraoperative and postoperative complications. Of the 120 patients under 40 years of age, only three had the ELC. Of the 102 patients above 40 years, 61 had the ELC. For decades above the third, 41 of the 50 patients who had CAD (82%) had the ELC, compared with 20 out of 52 non-CAD patients (38.5%) with the ELC.

For each decade beyond the third, intraoperative cardiovascular complications occurred more frequently than in the patients without the ELC. Forty-two percent (26) of the patients with the ELC developed intraoperative complications, in comparison to 4.9 or two of the 41 without the ELC. The complications included hypotension, hypertension and arrhythmias.

Postoperative cardiovascular complications were greater in those subjects with the ELC than those subjects without the ELC. Twenty-four percent (15 patients) of the ELC patients developed postoperative complications in comparison to 4.9% (2 patients) without the ELC. Overall, Sprague felt that the ELC could be used as a helpful indicator of intra- and postoperative complications, and that the ELC was not solely age-related.

The second study where the ELC was used as a screening tool was performed by Moncada, Ruiz, Rodriguez and Leiva (1979) in Mexico. They studied 300 apparently healthy individuals (factory workers, teachers and university students) and observed

for the ELC and data on certain risk factors for vascular disease, blood pressure readings, blood glucose, cholesterol and triglycerides, fundoscopy and electrocardiography. One hundred and fifty subjects with the ELC were observed with 150 age-matched controls. Thirty-five of the individuals with the ELC had hypertension compared with nine in the control group. Twenty individuals in the study group had eye ground changes in comparison to three in the control group. Electrocardiogram changes were apparent in 34 of the study group and in 11 of the control group. There were significant changes in blood glucose, triglycerides or cholesterol levels. The authors felt that apparently healthy persons with the ELC were more likely to have hypertension, abnormal fundoscopy or signs of cardiac ischemia on electrocardiogram than persons without the ELC.

#### Controversy in the Literature

Not all the literature is supportive of the suggestion that there exists a positive correlation between the ELC and CAD. Mehta and Hamby (1974) observed 211 consecutive patients undergoing coronary angiography. Coronary artery disease was found to be present in 159 patients, of whom 89 had the ELC (56%). Of the remaining 52 patients with normal coronary angiograms, 26 had the ELC (50%). The authors did not feel that the difference was statistically significant. However, they found a distinct increase in the presence of the ELC with advan-



cing age. The ELC was present in 65% of the patients over 55 years and 34% of those under 55 years. Mehta and Hamby (1974) suggested that it is age alone that is responsible for the ELC. It is observed that the control population (as in previous studies) was relatively small, and was also a selected group rather than a random one. Mehta and Hamby also selected just two groups, above or below age 55 years, which would appear to be an inadequate classification. Lichstein et al. (1974) previously suggested that the increased prevalence occurred between the age 50-59 age group. Frank (1973) in his initial observations in his small uncontrolled study, observed only those patients under 60 years of age, which excluded persons with advanced age, and yet the incidence of the ELC in CAD patients was high.

A second opinion disagreeing with the claim of correlation is offered by Haines (1978), who feels that its use as a diagnostic tool is a "myth which the New England Journal of Medicine continues to perpetuate" (p.1181).

Based upon data obtained from the National Center for Health Statistics, he states that the number of persons with CAD in the United States is approximately five percent of the population. Using a method described by Schwartz for data analysis, he felt that the results obtained by Lichstein et al. (1974) would render 92% false positive results. It was previously acknowledged by Lichstein et al. that their findings were not highly significant.

It is apparent that the research performed thus far with two exceptions, demonstrates a correlation between CAD and the ELC. There appears to be much disagreement as to whether or not the ELC is positively correlated with any or all of the risk factors for CAD.

#### Unknown Anthropological and Genetic Influences for CAD and the ELC

Previously in the introduction to this thesis, Williams (1980) was referred to for his research into the genetic aspects of CAD. His observations have been detailed, meticulously performed and lengthy. Review of his specific findings relative to CAD are beyond the scope of this endeavor; however, he is of the opinion that genetic factors are involved in a major way in the etiology of CHD, and that "greater knowledge of these genetic factors will improve the understanding of other issues involved in CHD" (1980, p. 89). Williams has not performed his studies with a view to supporting or not supporting the ELC and CAD suggestion, but he has included the presence or absence of an ELC in questionnaires used in his studies. The ELC information has yet to be compiled and analyzed due to the enormity of the venture, but the findings would have much bearing on this topic.

Kristensen and Peterson (1978) stated that the genetic factors predispose to hypertension and CHD, and that the C3<sup>f</sup> allele has been associated with atherosclerotic diseases such as

angina, claudication and myocardial infarction. The study performed, using 164 study individuals with hypertension and 80 control individuals without hypertension, investigated the occurrence of the C3<sub>f</sub> allele gene. In addition, the relative CAD risk factor was calculated using the Woolf method. At that time, the individuals in both groups were observed for the presence or absence of an ELC. Such creases were found in 19 out of 74 (Williams, 1980) male hypertensive patients (25%) and found in only one (3%) of the 29 normotensive males. Hypertensive females demonstrated 5 in 51 (8%) and 2 in 26 (7%). Kristensen and Peterson found the presence of the C3<sub>f</sub> allele in 73% of the patients with hypertension and there was an estimated ten-fold risk for CHD in these C3<sub>f</sub> positive individuals. Their report suggested a "hint" at a possible relationship with the C3<sub>f</sub> gene.

CHD is over three times as common in the United States as in Japan and it is suggested by Williams (1980) that genetic differences, as well as environmental differences, contribute greatly to the difference. Observation of hypercholesterolemic families in the United States, compared with Finnish families indicates distinct differences in genetic patterns.

Kaukola et al. (1979) cited Halonen as stating that the form of the branching of the coronary arteries and the vascularization of the myocardium differ between the Japanese and the Finns. Coronary artery disease is relatively infrequent

among the Japanese and so is the ELC (Kaukola et al., 1979; Rhoades et al., 1977).

The study by Schoenfeld et al. (1980) also included observations of Ashkenazi Jews and non-Ashkenazi Jews, MI and the ELC. Ear-lobe biopsy specimens at postmortem were included in that study and 12 specimens were obtained. Histologically, thickening of the prearteriole wall was found in some subjects with the ELC, with or without MI and in MI alone, but was not found at all in the non-MI or non-ELC subjects. Elastic fiber tears were found only in those subjects with the ELC. Due to the small samples, no conclusions were reached, but Schoenfeld speculated that a diminished blood supply to the highly vascular area might contribute to the elastic tears. It is noted, at this point, that the elastic element of the arteries of the Negro population manifest a lesser tendency to fragment and tear and that the incidence of CAD is lower in Negroes than in non-Negroes (Owen, Blach & Handler, 1950). A larger number of ELCs were found in the Ashkenazi Jews with MIs than the non-Ashkenazi Jews with MIs. The suggestion was made that the ELC may be an indicator of risk factors and be more prevalent among patients with diabetic retinopathy, hypertension and among persons of certain ethnic origin.

A study which observed the coronary arteries of three different ethnic groups were observed for histologic changes and compared (Vlodaver, Kahn & Neufeld, 1969). Necroscopy specimens

from Ashkenazi, Yemenite and Bedouin children were used. The Ashkenazi male was found to have more collagen tissue components, the intima was thicker and the musculoelastic tissue was more developed than the Ashkenazi female. These layers were far more developed in the Ashkenazi males than the Yemenites and Bedouin males. The intimal development of the Ashkenazy group is similar to American and European infants (Schornagel, 1956), while the intima of the Yemenite and Bedouin is similar to ethnic groups with a low incidence of CAD. The study of different ethnic groups, with and without ELC, would provide much information on this aspect of intimal thickness and perhaps, intimal tears.

Neufeld, Wagenvoort and Edwards (1962) in studies of the coronary arteries of children, showed that the external diameter, medial thickness and intimal thickness increase progressively with age; the male greater than the female. It is suggested that this increase in thickness and inevitable narrowing of the vessels would support the suggestion that advancing age, CAD and the ELC go hand in hand.

It is suggested that one or some unknown genetic and unknown anthropological factors responsible for CAD have some involvement also with the presence of the ELC. However, research in this area thus far has been limited, but it appears to be a promising avenue which needs to be explored.

### Summary

Consistently, the results of researchers reviewed indicated that some relationship exists between CAD and the ELC. However, as summarized in Table 6, there is a varying degree of significant correlation between the ELC and CAD risk factors. In light of this, the importance of prevention and possibility of positive correlation required further investigation.

### Research Questions

The following research questions were addressed in this investigation:

1. Does a relationship exist between CAD and the diagonal ELC?
2. Does a relationship exist between the risk factors for CAD and the ELC?
3. Do males demonstrate a greater prevalence for the ELC than females?
4. Does ethnic origin influence the prevalence of the ELC?

### Hypotheses

The following hypotheses were investigated:

1. A positive correlation exists between the presence of the ELC and the presence of CAD.
2. A positive correlation exists between the presence of the ELC and some of the risk factors for CAD (diabetes,

Table 6

Classification of Researcher Findings According to a Positive Correlation with the ELC

Researcher(s)	Year	CAD	Age	Hyper-tension	Smoking	Elevated Cholesterol	Diabetes	Type A Personality	Eye Changes	Obesity	Family History	Cardiovascular Complications
Anderson et al. (1976)		+	+									
Andreson et al. (1976)									+			
Doehring et al. (1977)		+	+									
Frank (1973)		+	+		+		+				+	
Haines (1978)												
Kaukola et al. (1978)		+	+									
Lichstein et al. (1974)		+	+									
Kristensen & Petersen (1978)		+		+								
Mehta & Hamby (1974)			+									
Moncada et al. (1979)		+							+			
Petrakis (1981)		+		+				+				
Rhoades et al. (1977)										+		
Shoenfeld et al. (1980)		+		+		+	+		+			
Sprague (1976)												+
Sternlieb et al. (1974)		+										

Note. + indicates positive correlation.

hypertension, obesity, cigarette smoking, stress, elevated cholesterol and heredity).

3. Males demonstrate greater prevalence of the ELC than females.

4. Caucasian, American culture increases the incidence of the ELC as opposed to any other culture.

### Definitions

#### Conceptual Definitions

CAD. Coronary artery disease (CAD) was defined as a diminished blood supply through the coronary arteries (the most common cause of which is atherosclerosis), which results in diminished blood supply and, therefore, a diminished supply of oxygen and nutrients to the heart muscle.

ELC. An ear-lobe crease was defined as a visible variation on the lobular portion of the ear which is a biological deviation from the norm. This visible variation is not found at birth, but develops with advancing age.

Risk factors for CAD. Risk factors for CAD were defined as acquired or inherited characteristics which a person possesses that place an individual at a greater risk for CAD. Major risk factors are those which are highly or strongly predictive of CAD. Minor risk factors are predictive or contributory but to a lesser degree. These risk factors include: advancing age, male sex, heredity, elevated cholesterol levels, hypertension,



diabetes, cigarette smoking, obesity, physical inactivity, and personality type-A.

Ethnic origin. The term ethnic origin was defined as the birthplace of the individual or the individual's parents which contributes to differences in habits, customs and beliefs of that individual.

#### Operational Definitions

CAD. Coronary artery disease (CAD) was defined as a) a reduction by over 50% in the lumen of one or more of the coronary vessels which results in diminished blood flow to the myocardium, as demonstrated by coronary angiography; and/or b) a reduction in the coronary blood flow which results in S-T changes on the electrocardiographs, Q wave changes on the electrocardiograph, and physical symptoms which result in a physician diagnosis of CAD, or myocardial infarction.

ELC. An ear-lobe crease was operationally defined as a deep crease or wrinkle which descends obliquely across the lobular portion of the ear-lobe. It is present unilaterally or bilaterally and extends for a distance equal to or greater than one third of the ear-lobe.

Risk factors for CAD. The following risk factors for CAD were operationally utilized and defined:

1. Advancing age: Individuals above the age of 30 years who are no longer in a biological growth period of their lives;

2. Male sex: The sex which has organs for producing sperm and for fertilization of ova. An individual of the male sex overall presents with: a deeper voice, male body and facial hair, less body fat, male genitalia and commonly known male-like qualities;

3. Heredity: The genetic influences which are transmitted from parents to offspring (contained in the chromosomes), which determine the inherited characteristics of the individual (e.g., skin color, hair, color, height, build);

4. Elevated cholesterol: A serum blood cholesterol level of above 250 milligrams per 100 milliliters of blood;

5. Hypertension: Blood pressure readings above those recommended by the American Heart Association for a given age group, i.e., 140/90 below age 55 years, and 160/90 for above age 55 years;

6. Diabetes: Fasting blood glucose levels above 120 milligrams per 100 milliliters, regardless of type of diabetes;

7. Cigarette smoking: The smoking, by an individual, of at least one cigarette daily;

8. Obesity: An abnormal amount of body fat which results in the individual being 20 percent or more above average weight for his/her sex and height;

9. Positive family history: A member of the immediate family; i.e., parent sibling and/or grandparent sustained an early myocardial infarction; males before age 60 and females

before age 50.

10. Personality Type A: Individual traits and characteristics which render the individual competitive, impulsive, compulsive, easily angered, achievement oriented, and aggressive; as described by Friedman and Rosenman (1979).

#### Assumptions

The following assumptions influenced the current investigation:

1. Identification of the ELC as an indicator of possible CAD would be a simple, inexpensive, noninvasive tool in CAD detection.
2. Early identification of CAD would increase the likelihood that lifestyle and risk factors might be modified.
3. An earlier reduction in the risk factors for CAD might reduce morbidity and mortality for CAD.
4. Society as a whole would benefit by having a readily available, nonintrusive tool for the identification of CAD.

## CHAPTER III

### METHODOLOGY

#### Design

The research design used was a descriptive, cross-sectional, ex post facto approach to study ear-lobe crease and coronary artery disease (CAD). There was no manipulation of the variables, the collection of data was obtained at one time only, and there was no observation of the subjects over a period of time. A study group and a control group were observed, and the method of reasoning was inductive.

#### Setting

The data collection was performed at Holy Cross Hospital, Salt Lake City, Utah (343 beds) which is a Catholic, nonprofit hospital situated in an urban area in the mountain west region of the United States. This hospital admits just over 1000 individuals per year with a diagnosis of CAD and/or myocardial infarction (MI).

#### Sample

The population was selected from a cluster sample, and the study group included all individuals, both male and female, above the age of 30 years admitted to an urban hospital with a

physician diagnosis or angiographically demonstrated diagnosis of CAD and/or MI. The control group, also randomly selected, was age-matched to within the same decade, and sex-matched with the study group, but without the above mentioned criteria of CAD and MI. The sample included 77 subjects in the study group and 77 subjects in the control group.

### Instruments

A two-part, three page data collection sheet was devised and was used by the study group participants and the control group participants to document personal information. A second, one page data sheet was developed and used to condense that information and to document the presence of an ELC.

### Procedure

Permission to conduct this research study was obtained from the Human Subjects Committee of the University of Utah and the Human Subjects Committee of the investigational site. Upon approval from these committees, the principal investigator met with medical physicians (primarily cardiologists) to explain the study, the rationale for the study and obtain their individual permissions. A similar meeting was held with the supervisors of the involved nursing units and the nursing personnel to explain the purpose of the study.

### Data Collection

A list of patient admissions, which included name, age and diagnosis was obtained from the admissions office on a daily basis. Study group subjects and control group subjects read or had read to them the patient informed consent form and verbal and written agreement to participate in the study was obtained. Each participant completed, or was assisted in completing, the personal information data collection sheet.

Study group subjects and control group subjects were observed for the presence or absence of a unilateral or bilateral ELC. This observation was performed by the principal investigator or a research assistant. Upon observation of the ear-lobe, the investigator completed the data collection sheet from information obtained from the personal information sheet and the participant's hospital chart.

Threats to internal validity of the study were reduced by:

1. No treatment being involved;
  2. Not including history, maturation, testing and mortality as factors.
  3. Randomization and the use of a comparison control group.
  4. Maintaining constancy of conditions, i.e., same observer, same criteria, same sex and same age group.
  5. Testing interrater reliability prior to the study.
- This was accomplished by concurrent principal investigator and

research assistant observation of at least ten ear-lobes with 100% agreement.

Threats to external validity were reduced by:

1. Not involving interactive, reactive, novelty or Hawthorne effects.
2. Using collaborative agreement between the principal investigator and the research assistant when there was doubt regarding the presence or absence of the ELC to avoid experimental effects.

#### Human Subjects Considerations

The observation of the subjects ear-lobes occurs within the realm of the routine physical assessment performed by all members of the nursing staff. Therefore, no additional procedures were required. The observation did not require the ear-lobe to be touched by the investigator, as the ELC is readily visible to the naked eye.

The information contained within the subject's chart is information which is already accessible to the investigator without invasion of privacy; however, an Informed Consent (Appendix E) was used to inform the subjects that the information would be gathered and to request their permission to be part of the study. The following information was given to them:

1. A full explanation of the study and possible implications.

2. Study participation was voluntary and could be withdrawn at any time.

3. An identification number was given to maintain confidentiality and prevent invasion of privacy. The master key to the information was protected from access by other individuals.

4. There were no known physical or discomfort risks involved.

5. There was no deception used.

6. There were no known moral, ethical, or value issues involved.

#### Risks and Potential Benefits

A potential emotional risk was involved when a subject had an ELC and was made aware of the possible implications, especially if CAD had not been previously diagnosed.

With the ELC identified as a possible indicator of CAD, earlier identification of CAD would be facilitated. Earlier identification might produce an earlier modification of lifestyle and possibly reduce the morbidity and mortality of CAD. A benefit for the individual and society as a whole would ensue.



## CHAPTER IV

### ANALYSIS OF THE DATA

#### Demographic Information

The sample consisted of 154 voluntary participants selected from a population of patients admitted to Holy Cross Hospital during the period June 1982 to June 1983. There were 69 males in the control group as in the study group and eight females in both groups, as well.

The age of the study group subjects ranged between 32 and 89, with the mean age of the sample being 61.5. The age of the control group subjects ranged between 33 and 83, with the mean age of the sample being 61.44 (Table 7).

The groups were cross-matched for sex and age to within a decade. All participants in the study were Caucasian Americans, with no further limitation required as to area or place of origin. The majority of participants, however, resided in and/or came from the Salt Lake City vicinity.

#### Results

The statistical programs utilized for data analysis were

Table 7  
Demographic Characteristics of the Sample

Characteristic	Study Group	Control Group
Sex		
% Male	95	95
% Female	5	5
Total	100	100
Age in Years	<u>N</u>	<u>N</u>
30-39	4	4
40-49	14	14
50-59	4	4
60-69	36	36
70-79	16	16
80-89	3	3
Range	32-89	33-83
Mean	61.5	61.4
<u>n</u>	77	77
% Caucasians	100	100

a) condescriptive, to establish the range and maximum and minimum, values, b) crosstabulations, to establish all tables and statistics, c) corrected chi-square to establish a relationship of d) Phi, Cramer's and contingency coefficient tests, to establish the strength of that relationship, and e) Fisher's Exact Test was used for  $N_s$  of 5 or below. Significance was established at  $p < .05$  for all correlation and chi-square tests.

### Research Questions

#### Research Question One

The first research question identified during the study examined the relationship between Coronary Artery Disease (CAD) and the diagonal ear-lobe crease (ELC):

Does a relationship exist between CAD and the diagonal ELC?

Seventy-four individuals with documented myocardial infarctions (MIs) were observed for the presence of an ELC and the control group of 77, who had not sustained an MI, crossmatched for age and sex, was also observed. Of the 77 individuals in the study group, 75 percent (58 individuals) had an ELC present, 25 percent (19 individuals) did not. Of the 77 individuals in the control group, 35 percent (28 individuals) had an ELC and 64 percent (or 49 individuals) did not. The majority of those individuals who demonstrated the ELC had one bilaterally. Of those who had a unilateral ELC, it occurred predominantly on the right ear-lobe in the study group (9:3) and

in the left ear-lobe in the control group (5:2).

As indicated in Table 8, corrected chi-square demonstrated a highly significant relationship of  $p < .0001$  between CAD and the presence of the ELC.

#### Research Question Two

The second question posed looked at the relationship between the ELC and some of the risk factors for CAD. The risk factors observed were sex, age, and heredity, which are considered nonmodifiable, and cigarette smoking, hypertension, elevated serum glucose, elevated serum cholesterol, stress and obesity which are considered modifiable.

Does a relationship exist between some of the risk factors for CAD and the presence of the ELC?

Sex and the ear-lobe crease. The number of males in both groups far exceeded the number of females (69:8) and, therefore, made a statistical comparison of the sexes unacceptable. A crosstabulation of the ear-lobe crease (ELC) and sex was performed and it revealed that 78 males and 8 females had the ELC and 60 males and 8 females did not have the ELC (total of 154 subjects). The findings demonstrated no correlation between the sex of the individual and the presence or absence of an ELC as indicated in Table 9.

The male study group had 55 males with an ELC and 14 who did not have a crease; while the control group had 24 males who did have an ELC and 45 who did not. A chi-square analysis was

Table 8  
Coronary Artery Disease and the Ear-Lobe Crease

	Study Group (Myocardial Infarc- tion Patients) <u>N</u>	Control Group (Nonmyocardial Infarction Pa- tients) <u>N</u>
With ELC	58 (75.3%)	28 (36.4%)
Without ELC	19 (24.7%)	49 (63.6%)
Total <u>N</u>	77 (100.0)	77 (100.0%)
ELC/CAD Correlation positive Chi-square = $\chi^2$ = .0000** Phi = .3878 Contingency coefficient = .3616		

Note. \*\*Highly significant,  $p < .0001$ .

Table 9  
Relationship Between Sex and the Ear-Lobe Crease

Variable	Study Group N	Control Group N
Males		
ELC	55 (79.7%)	24 (34.8%)
No ELC	14 (20.3%)	45 (65.2%)
<u>n</u>	69	69
Females		
ELC	4 (50.0%)	4 (50.0%)
No ELC	4 (50.0%)	4 (50.0%)
<u>n</u>	8	8
Correlations		
	Chi-square <u>p</u> =	Phi Contingency Coefficient
Sex and ELC	.6266	No correlation
ELC/CAD with male sex controlled positive	.0000**	.4466 .4078
ELC/CAD with female sex controlled	--	.0714 .0716
	(Fisher's exact test = .5952)	

Note. \*\*highly significant;  $p < .001$ .

performed on this data, controlling for the male sex through the use of a stratified contingency table analysis. As shown in Table 9, a strong correlation of  $p < .0001$  between the ELC and CAD was demonstrated.

The number of females in both groups was eight, a small sample size, and both groups had equal numbers of subjects with and without the ELC; four of each for both groups. In view of the equal numbers in all cells and the low numbers involved, chi-square analysis was not performed and a Fisher's exact test was performed instead. As indicated in Table 9, the Fisher's test, at .5952 was not significant ( $p = .07$ ).

Heredity and the ear-lobe crease. The ear-lobe crease (ELC) and heredity for coronary artery disease (CAD) were cross tabulated for a correlation and no correlation was found between the two. Out of 67 individuals without the ELC, 26 had a history of CAD in their immediate families and 41 did not. The study group demonstrated almost equal numbers of those with a positive heredity component: 44 without family history and 43 with.

When the lack of hereditary influence was controlled for, the correlation between CAD and the ELC statistically was highly significant at the  $p = .0001$  level (Table 10). Likewise, when the presence of hereditary influence was controlled for, the results were equally supportive of a CAD/ELC correlation (Table

10).

Age and the ear-lobe crease. As previously stated, the age range of the study group was 32 to 89, with a mean of 61.5. The range of the control group was 33 to 83, with a mean of 61.4. The age data from both groups was divided up into decades, starting at age 30-39 and finishing at age 80-89. Each age group was crosstabulated for a correlation with the ELC and a positive correlation of  $p = .0169$  was found as demonstrated in Table 11.

When each age group was controlled for, a positive correlation was demonstrated between the ELC and CAD in some age groups but not others (Table 12, Figure 2)(See Table 13 for a summary of nonmodifiable risk factors and CAD).

Cigarette smokers and the ear-lobe crease. A crosstabulation of both groups combined revealed a total of 59 smokers, 68 without an ELC and 86 with an ELC. Of the 68 without an ELC, 40 were nonsmokers and 28 were smokers. Analysis of the data demonstrated no correlation between cigarette smoking and the ELC as shown in Table 14.

Further observations of the data, with the smoker aspect being controlled for, revealed a positive correlation of statistical significance between the ELC and CAD as indicated in Table 14. With the nonsmoker aspect being controlled for, a positive correlation of statistical significance between CAD and the ELC was also demonstrated (see Table 14).



Table 10  
Family History of CAD and the Ear-Lobe Crease

	Positive Family History for CAD		Negative Family History for CAD
ELC	43		44
No ELC	26		41
Correlations			
	Chi-square	Phi	Contingency Coefficient
Family History for CAD and the ELC	No correlation found ( $p = .2500$ )		
ELC and CAD without hereditary influence controlled	.0022*	.3556	.3351
ELC and CAD with hereditary influence controlled	.0017*	.4094	.3789

Note. \* $p < .05$ ; significant.

Table 11  
Control and Study Group Ages and the Ear-Lobe Crease

Age	<u>N</u>	ELC Present <u>N</u>	% ELC Present
Study Group			
30-39	4	2	50.0
40-49	14	7	50.0
50-59	4	3	75.0
60-69	36	32	86.5
70-79	16	12	75.0
80-89	3	3	100.0
Total	77		
Control Group			
30-39	4	--	--
40-49	14	3	21.4
50-59	4	--	--
60-69	36	17	48.6
70-79	16	8	50.0
80-89	3	--	--
Total	77		

Note. Study group and age showed positive correlation.  
 $p = .0169$

Table 12  
Age and the Ear-Lobe Crease

	30-39	40-49	50-59	60-69	70-79	80-89	<u>N</u>
No ELC	6	18	5	23	12	3	67
ELC	2	10	3	49	20	3	87
Total	8	28	8	72	32	6	154

Note. Chi = .0169.

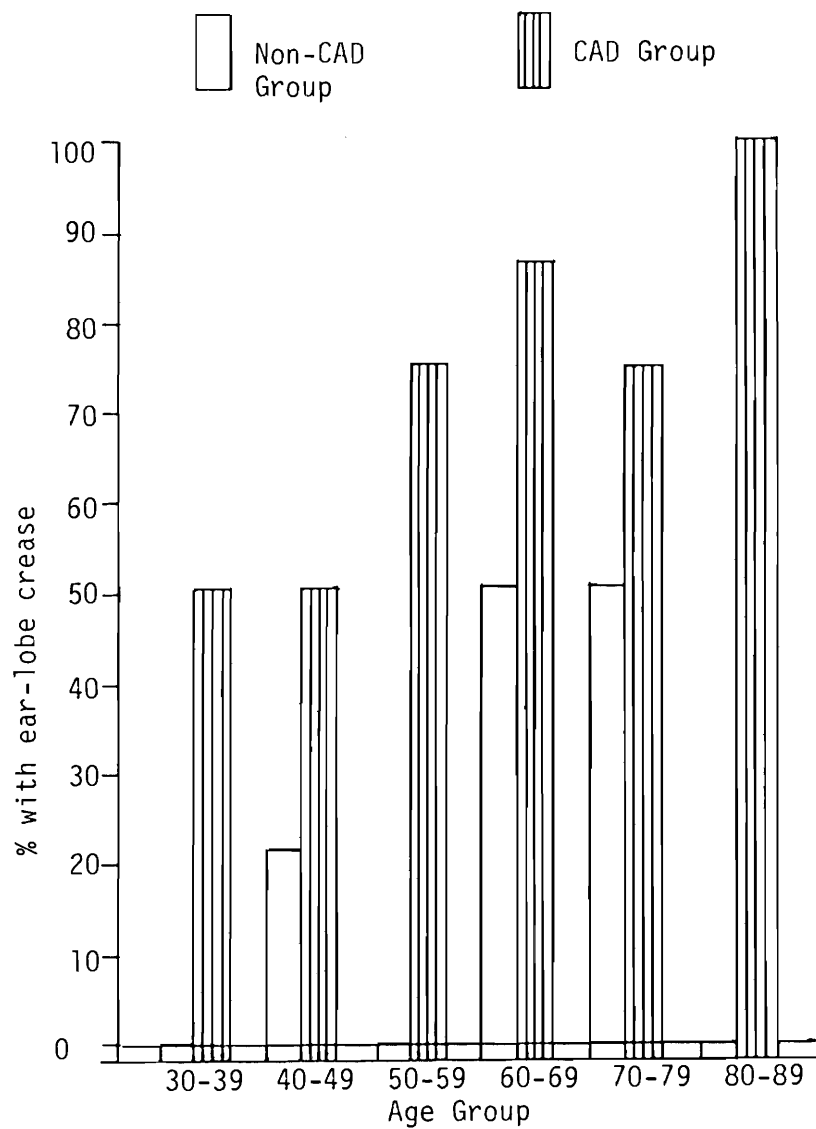


Figure 2. Relationship of Coronary artery disease (CAD), Age and the diagonal ear-lobe crease.

Table 13  
 Presence of an ELC in CAD Subjects and Non-CAD Subjects  
 According to Nonmodifiable Risk Factors

	CAD Group <u>N</u>	Non-CAD Group <u>N</u>
Males	55 (79.7%)	24 (34.8%)
Females	4 (100.0%)	4 (100.0%)
Age		
30-39	2 (50.0%)	0 (0.0%)
40-49	7 (50.0%)	3 (21.0%)
50-59	3 (75.0%)	0 (0.0%)
60-69	32 (86.5%)	17 (48.6%)
70-79	12 (75.0%)	8 (50.0%)
80-89	3 (100.0%)	0 (0.0%)
Positive Family History	43 (49.4%)	26 (38.8%)

Table 14  
Cigarette Smokers and the Ear-Lobe Crease

	Smokers		Non-Smokers
ELC	32 (37.2%)		54 (62.8%)
No ELC	28 (41.2%)	<u>n=86</u>	40 (58.8%)
		<u>n=68</u>	
Correlations			
	Chi-square	Phi	Contingency Coefficient
Cigarette smokers and the ELC	No Correlation Found ( <u>p</u> = .4294)		
ELC and CAD with smoking not controlled	.002*	.3407	.3225
ELC and CAD with smoking controlled	.0005*	.4860	.4371

Note. \* $p < .05$  (significant).

Elevated serum glucose and the ear-lobe crease. There were a total of 12 subjects who had a history of elevated blood glucose and these were divided equally between those subjects who had the ELC and those who did not; six in each group. This meant that of the 68 subjects without the ELC, 62 did not have elevated serum glucose and of the 86 subjects with the ELC, 80 did not have an elevated serum glucose. Analysis of the data demonstrated no correlation between elevated serum glucose and the ELC (Table 15).

CAD and its relationship to the ELC was analyzed, controlling for elevated serum glucose and for nonelevated serum glucose. When the absence of an elevated serum glucose was controlled for, highly significant data was obtained (see Table 15). When evaluating and controlling for the presence of serum glucose elevation the small sample (6 in both the control group and the study group) determined that chi-square testing would not be valid due to the low expected cell frequency number. However, Fisher's exact test was performed and demonstrated a significant correlation between CAD and the ELC when elevated serum glucose was controlled for (Table 15).

Hypertension and the ear-lobe crease. Hypertension and the ELC were crosstabulated to assess for a positive correlation and a positive correlation was demonstrated. Of the 86 with an ELC present, 34 had hypertension and 52 did not. Of the 68 without an ELC present, 15 had hypertension and 53 did not (see Table

Table 15  
Serum Glucose and the Ear-Lobe Crease

	Elevated Serum Glucose N	Elevated Serum Glucose N	
ELC	6 (7.0%)	80 (80.0%)	
	<u>n</u> =86		
No ELC	6 (8.8%)	62 (91.2%)	
	<u>n</u> =68		
Correlations			
	Chi-square	Phi	Contingency Coefficient
Elevated serum glucose and the ELC	No correlation found ( <u>p</u> =.8819)		
ELC and CAD with absence of ele- vated serum glucose controlled	.0000**	.3461	.3421
ELC and CAD with the presence of elevated serum glucose Fishers Exact Test = .0400	--	.6667	.5547

Note. \*\*p < .0001 (highly significant)



15).

When the absence of hypertension was controlled for, and the ELC and CAD were evaluated for a correlation, a positive correlation was also found (Table 16). Similarly, when the presence of hypertension was controlled for and the ELC and CAD were compared, a positive correlation was also found (Table 16).

Stress and the ear-lobe crease. The occurrence of stress and the presence of the ELC were crosstabulated to demonstrate a positive correlation, however; none was found. Of the 68 individuals without the ELC, 27 did not admit to being of the A type of personality and 41 did. Individuals with the ELC, 86 in all, were almost evenly divided between those of type A personality (46 individuals) and those of Type B personality (40 individuals). Corrected chi-square did not demonstrate a positive correlation between stress and the ELC (Table 17).

With the absence of stress controlled for, CAD and the ELC demonstrated highly significant results for a correlation (Table 17). The presence of stress was then controlled and CAD and the ELC again demonstrated highly significant data.

Obesity and the ear-lobe crease. The ELC and its relationship to obesity data were analyzed and no relationship was demonstrated. Of the 64 individuals without an ELC, 43 were not obese and 21 were (4 with missing data). Twenty-three of the 86 individuals with an ELC were obese and 63 of them were not (Table 18).

Table 16  
Hypertension and the Ear-Lobe Crease

	Hypertension	No Hypertension	
	<u>N</u>	<u>N</u>	
ELC	34.0 (39.5%)	52.0 (60.5%)	
	<u>n</u> = 86		
No ELC	15.0 (22.0%)	53.0 (78.0%)	
	<u>n</u> = 68		
Correlations			
	Chi-square	Phi	Contingency Coefficient
Hypertension and the ELC	.0220*	(positive correlation)	
ELC and CAD with absence of hypertension controlled	.0024*	.3155	.3009
ELC and CAD with presence of hypertension controlled	.0026*	.4832	.4351

Note. \* $p < .05$  (significant)

Table 17  
Stress and the Ear-Lobe Crease

	Stress <u>N</u>	No Stress <u>N</u>	
ELC	46.0 (53.5%)	40.0 (46.5%)	
	<u>n</u> = 86		
No ELC	41.0 (60.3%)	27.0 (39.7%)	
	<u>n</u> = 68		
Correlations			
	Chi-square	Phi	Contingency Coefficient
Stress and ELC	No correlation found ( <u>p</u> = .4294)		
ELC and CAD with absence of stress controlled	.0002*	.4869	.4376
ELC and CAD with presence of stress controlled	.0042*	.3303	.3137

Note. \*p < .05 (significant ).

Table 18  
Obesity and the Ear-Lobe Crease

	Obesity <u>N</u>	No Obesity <u>N</u>	
ELC	23.0 (26.7%)	63.0 (73.3%)	
	<u>n</u> = 86		
No ELC	21.0 (32.8%)	43.0 (67.2%)	
	<u>n</u> = 64		
Correlations			
	Chi-Square	Phi	Contingency Coefficient
Obesity and ELC	No correlation ( <u>p</u> = .5313)		
ELC and CAD with absence of obesity controlled	.0007*	.3495	.3299
ELC and CAD with presence of obesity controlled	.0069*	.4530	.4127
Note. *p < .05 (significant)			

Absence of obesity was then controlled for and the ELC and CAD relationship was observed, demonstrating a strong positive correlation (Table 18). Similarly, when the presence of obesity was controlled for, again the strong correlation between the ELC and CAD was demonstrated (Table 18).

Cholesterol and the ear-lobe crease. Elevated cholesterol and the ELC were crosstabulated for a relationship and some correlation was found. One hundred and fifty three subjects' (one with missing data) data revealed that of 67 with no ELC, five had elevated cholesterol and of the 86 subjects with the ELC, 69 had no elevated cholesterol and 17 of them had an elevated level (see Table 19).

Controlling for the absence of elevated cholesterol, the correlation between the ELC and CAD demonstrated a positive correlation with highly significant results (Table 19). Elevated cholesterol was controlled for and the positive correlation between the ELC and CAD again was highly significant (see Table 20 and 21 for summary of modifiable risk factors).

### Research Question Three

The third research question was one that this study was unable to address:

Do males demonstrate a greater prevalence for the ear-lobe crease than females?

The number of males in each group was 78 and the number of females 8, therefore, the inequity of numbers made any attempts

Table 19  
Elevated Cholesterol and the Ear-Lobe Crease

	Elevated Cholesterol <u>N</u>	Normal Cholesterol <u>N</u>	
ELC	17.0 (19.8%)	69.0 (80.2%)	
<u>n</u> = 86			
No ELC	5.0 (7.5%)	62.0 (92.5%)	
Correlations			
	Chi-square	Phi	Contingency Coefficient
Elevated Cholesterol and ELC	.0314*	(positive correlation)	
ELC and CAD with absence of elevated cholesterol controlled	.0049*	.3190	.3039
ELC and CAD with presence of elevated cholesterol controlled	.0041*	.7412	.5955

Note. \* $p < .05$  (significant).

Table 20  
 Presence of an ELC in CAD Subjects and Non-CAD Subjects  
 According to Modifiable Risk Factors

	CAD Group <u>N</u>	Non-CAD Group <u>N</u>
Smokers	32.0	28.0
Elevated serum glucose	6.0	6.0
Hypertension	34.0	15.0
Stress	46.0	41.0
Elevated serum cholesterol	17.0	5.0
Obesity	23.0	21.0

Table 21  
Correlations of Study Variables and the Ear-Lobe Crease

Variable	Correlation	Significance Level
CAD	Positive	.0000**
Sex	None	.6226
Age	Positive	.0169 *
Heredity	None	.2500
Smokers	None	.4294
Hypertension	Positive	.0220*
Stress	None	.4294
Elevated glucose	None	.8819
Elevated cholesterol	Positive	.0314*
Obesity	None	.5313

Note. \*\* $p < .0001$ ; \* $p < .05$ .



to demonstrate any prevalence by males to the ELC statistically unsound.

#### Research Question Four

The fourth research question was also one that this study was not able to address:

Does ethnic origin influence the prevalence of the ELC?  
In attempting to answer this question, the study was hindered by the small number of non-Caucasians available to include in the sample. Salt Lake City is predominantly Caucasian, with the black population comprising just 1.5 percent and the hispanic population just 7.5 percent. In view of this, no further attempt was made to include this question in the analysis.

## CHAPTER V

### DISCUSSION

Present literature and previous studies both support and disagree with the hypothesis that there is a connection between the presence of an ELC and CAD. As indicated earlier, the previous studies had many weaknesses which this study attempted to control for. The tests of statistical significance used in this study clearly support that the relationship appears to be actual and does not occur through chance. Acknowledgment is given, however, to the fact that "there always remains a possibility that the relationships were due to chance" (Polit & Hungler, 1978, p.601).

The percentage of individuals with an ELC in this study group was 75 percent compared with 36 percent in the control group. Previous studies performed have varied with the percentage of individuals with the ELC in both the study and the control groups. As Table 22 indicates, percentages of previous study group individuals with the ELC varied from 47 to 94 percent and the control group individuals with the ELC varied

Table 22  
A Comparison of Individuals with the ELC

	Study Group %	Control Group %
Lichstein et al. (1973)	47.0	30.0
Doering et al. (1979a)	88.0	36.8
(1979b)	94.4	44.4
Shoenfeld et al. (1980)	77.0	40.0
Sprague (1976)	82.0	38.5
Kaukola et al. (1976a)	69.0	24.0
(1976b)	72.0	Unknown
Mehta et al. (1974)	56.0	50.0
Present study (1983)	75.0	36.0

from 47 to 94 percent and the control group individuals with the ELC varied from 24 percent to 50 percent.

All the studies where reliability factors gave  $p < .05$  or  $p < .0001$  factors when comparing the ELC and CAD; this study adopted the same reliability factors.

When all the risk factors for CAD were controlled for in this study, regardless of whether or not they themselves correlated, there was a positive correlation demonstrated between CAD and the ELC. Lichstein et al. (1973) observed hypertension, diabetes and smoking habits and controlled for these adequately, also resulting in a positive correlation ( $p < .0001$ ). Kaukola et al. (1976) also observed and controlled for the risk factors for CAD and also found a positive correlation.

Each individual included in the study group had, as previously stated, sustained one or more myocardial infarctions (MIs). The majority of the previous studies cited also had as study participants, individuals who had sustained MIs. It is of interest to note that the two authors who did not agree that there was a correlation between the ELC and CAD (Mehta & Hamby, 1974; Rhoades et al., 1977) did not state they had used subjects who had actually sustained an MI.

Mehta and Hamby did not find a correlation between the ELC and CAD but speculation arose concerning the authors' selection of the control group. They observed 211 individuals, and found

CAD in 159 patients, the remainder having normal angiograms. It is doubtful, ethically, that the authors performed angiography on asymptomatic individuals and that angiography was performed for some reason. This limits the ability of the control group to actually provide the controls necessary.

#### Risk Factors for CAD and the ELC

Nine risk factors were observed and out of these a positive correlation with the ELC was demonstrated by three of them; hypertension, elevated serum cholesterol and age. The significance levels established for age and hypertension were greater than those established for elevated serum cholesterol. No correlation was found between elevated serum glucose, stress, smoking, sex, heredity, obesity and the ELC.

#### Sex and the ELC

As previously stated, one of the original hypotheses of this study was that males have a greater prevalence for the ELC than females, but the number of females involved in this study was too small to make a comparison between the two groups (69:8). This was not the case with a few of the previous studies performed, for the number of females involved was adequate; however, in the majority of the studies the male/female breakdown was not mentioned. One wonders whether the lower number of females sustaining myocardial infarctions in Utah is due to the cultural influence of the LDS religious

health code, a question that warrants further study. Doering et al. (1977) observed 30 males and 20 females. Anderson et al. (1975) observed 56 males and 56 females, and Kaukola et al. (1976) observed 165 males and 54 females. Rhoades et al. (1977) did not observe any females but studied 1237 middle-aged Japanese men only.

This study found no correlation between sex and the ELC and the same was found in the one study that did include this in the observations (Shoenfeld et al., 1980). Surprisingly, Kaukola et al. (1976) did not include this in the evaluation of risk factors and yet the remainder of the study was so inclusive. It is questionable whether Kaukola felt this was not a factor or it was merely overlooked in the reporting.

The nature of the disease (CAD) at this point dictates that men are more prone to CAD than women before the menopause. It is unfortunate that the number of female subjects was low; a comparison of the two might have proved beneficial.

#### Heredity and the ELC

The hereditary aspect was not studied a great deal in previous studies, just twice in addition to the initial observation by Frank (1973). The studies that were performed were two of the most inclusive of all, those by Kaukola et al. (1976) and Shoenfeld et al. (1980). Both of these studies, as this investigation, found no correlation between the ELC and

a positive family history for CAD. Further observations are necessary of the hereditary aspect, especially investigations concerning the genetic aspect of the ELC and CAD.

#### Age and the ELC

Age has been demonstrated in six studies out of eight to have a positive relationship with the presence of an ELC. Anderson et al. (1975) and Shoenfeld et al. (1980) were the two partial dissenters, finding "some" correlation in the age groups beyond 50 years. No details of controlling for age with the ELC and CAD were outlined, so it is possible that these two studies also indicate a correlation but the details are unclear.

This study (see Table 23) demonstrated a correlation between age and the ELC. When controlled for age groups, two groups did not demonstrate a correlation between CAD and the ELC, ages 40-49 and 70-79. The reason for this remains unclear.

Advancing age appears to be the only one out of the three nonmodifiable risk factors for CAD that has a positive correlation with the ELC.

#### Cigarette Smoking and the ELC

No correlation was demonstrated between the ELC and cigarette smoking and this was also the consensus of all the studies that included cigarette smoking in their observations. Although listed as a risk factor for CAD, the effects of smoking are felt to be removed when the individual stops

Table 23

CAD/ELC Correlation with Age Controlled For

Age	<u>N</u>	Chi-square	Phi	Contingency Coefficient	Fishers Exact Test
30-39	4	--	.5774	.5000	.2143
40-49	14	.2367	.2981	.2857	--
50-59	4	--	.7746	.6124	.0714
60-69	36	.0014*	.4064	.3765	--
70-79	16	.2733	.2582	.2500	--
80-89	3	--	1.0000	.7071	.0500*

Note  $p < .05$  (significant).



smoking; the vasoconstrictive effects of nicotine being reversed upon cessation. It is suggested that, if the ELC is caused by intimal damage, smoking might contribute to the damage, but this connection was not supported statistically in this study.

Lichstein et al. (1973), Anderson et al. (1975), Shoenfeld et. al (198) and Kaukola et al. (1976) found no correlation between smoking and the ELC. However, Lichstein stated that the number of cigarette smokers was greater in the study group than the control group (68 percent to 56 percent). This difference was not noted in this study, with 41 percent of the control group and 37 percent of the study group as smokers.

#### Elevated Serum Glucose and the ELC

When Frank (1973) first suggested a correlation between the ELC and CAD, he suggested that diabetes mellitus might also be implicated. At that time, Lichstein et al. (1973), followed by Anderson et al. (1975) also examined for a relationship between the two and no relationship was found. The results of this study also demonstrate no support of a correlation, which conflicts with the results obtained by Shoenfeld et al. (1980) who felt that a relationship, albeit a weak one, exists between the two.

Andreson et al. (1976) examined 101 individuals with diabetes and examined them solely for retinal angiopathy in relationship to the ELC; a relationship was found. The suggestion that a relationship exists between coronary

angiopathy, retinal angiopathy and the ELC is an intriguing one, especially in view of the findings of no correlation between the ELC and diabetes.

#### Hypertension and the ELC

Hypertension has long been implicated in disease processes of the heart and its adverse effects in CAD are clearly established. Suggestion is made that it is involved in the process of the formation of the ELC, the exact mechanism being through intimal injury.

The results of this study clearly depicted, statistically, that a correlation exists between the ELC and the presence of an elevated blood pressure. A number of the previous studies performed also suggested a positive relationship, yet the results were inconclusive. Lichstein et al. (1973) in their early study did not demonstrate a correlation; Kaukola (1976), in her extensive study did not demonstrate a correlation nor did Anderson et al. in 1975. Each of these studies had an acceptable number of participants and the selection methods were appropriate. Conversely, Frank's (1973) original observations implicated hypertension as a "significant finding," but the study number was small and without a control group. Shoenfeld et al. (1980), like Kaukola, performed an extensive study involving 421 controls and 421 study individuals who were sex, age and ethnic origin matched; and yet unlike Kaukola et al., a

correlation was demonstrated between hypertension and the ELC.

Moncado et al. (1979) and Petrakis (1974) approached the correlation question differently; Moncado using the ELC as a screening tool, and Petrakis (1981) merely reporting on observations of busts of the Emperor Hadrian. Both felt that a correlation between the ELC and hypertension existed but Petrakis understandably supplied no supporting evidence.

Rhoades et al. (1977), found only a weak correlation between the ELC and hypertension, along with their findings of no relationship between the ELC and CAD.

Kristensen and Peterson (1980), when observing genetic factors that predispose to coronary heart disease (CHD) observed for the ELC in the hypertensive patients observed. They found a correlation between hypertension and the ELC in males only, but suggested a "hint" of a relationship between the ELC, CHF and the C3<sup>f</sup> gene.

One of the interrelated concepts fundamental to the suggestion that there is a positive correlation between the ELC and CAD is that the crease may be the result of the pathological process of the small vessels found in the presence of hypertension. With it postulated that the ELC is a response to injury, such as occurs within the hypertensive vessels, the demonstration of a positive correlation between hypertension and the ELC would, indeed, support the suggestion of injury as a precursor of the ELC.

It is interesting to note that no relationship was found by Shoenfeld et al. between hypertensive retinopathy and the ELC, yet Andresen et al. found that a correlation exists between the ELC and diabetic retinopathy. This leads to some question as to the relationship, if any, between hypertensive angiopathy, diabetic angiopathy and generalized angiopathy.

### Stress and the ELC

Previous studies did not evaluate a connection between the ELC and stress; however, this study did and no correlation was demonstrated. An isolated observation came from Petrakis (1980) who suggested a correlation, in view of Emperor Hadrian's aggressive personality, but supplied only "literary and sculptural evidence" to support that suggestion. At this point there is no support of a relationship between the two.

### Obesity and the ELC

Obesity, like stress, was not investigated to a great extent in previous studies. Kaukola et al. (1976) and Rhoades et al. (1977) both observed obesity and the ELC and obtained diversified results. Rhoades found a strong correlation, yet Kaukola found no correlation at all; similarly, no connection was found in this study. Obesity is not recognized as a risk factor for CAD per se, but is included because of the influence it exerts through increased hypertension and diabetes.

### Elevated Cholesterol and the ELC

Like hypertension, cholesterol has long been implicated in the atherosclerotic process. In view of this, the demonstration of a correlation between the ELC and CAD is significant. This study demonstrated such support, and although the studies observing elevated cholesterol were few in number, opinions varied. Moncado et al. (1979) and Shoenfeld et al. (1980) found a positive correlation, whereas, Kaukola et al. (1976) did not. Along with hypertension, elevated cholesterol is postulated to be involved in the process of ELC formation through its increased synthesis; a positive correlation lends more credence to this postulation.

### Risk Factors for CAD and the ELC

This study demonstrated a relationship between CAD, age, hypertension, and elevated cholesterol levels. On evaluating previous studies, age, CAD and hypertension were also the three factors where a positive correlation was most demonstrated (see Table 24).

### Conclusion

In conclusion, the present study clearly shows that a correlation exists between the ELC and CAD, age, hypertension and elevated cholesterol levels. Corrected chi-square with Phi, Cramer's and Contingency correlation coefficients (Nie, Hull, Jenkins, Steinbrenner & Bent, 1975) were used to demonstrate

Table 24  
Present Study Findings and Previous Study Findings

Investigator(s)	CAD	Age	Hyper- tension	Elevated Cholesterol
Present study	+	+	+	+
Anderson et al.	+	-	-	0
Doering et al.	+	—	0	0
Frank	+	+	+	0
Kaukola et al.	+	—	-	-
Lichstein et al.	+	—	-	0
Kristensen & Peterson	+	0	+	0
Mehta & Hamby		+		
Petrakis	+	-	+	0
Shoenfeld et al.	+	-	+	+
Sternlieb et al.	+	0	0	0

Note. 0 = not tested; + = positive correlation;  
- = negative correlation.

strength of any demonstrated correlation. The results indicated that the presence of the ELC demonstrated a statistically significant (positive) correlation with CAD, age, hypertension, and elevated serum cholesterol levels. There was no correlation with sex, heredity, elevated serum glucose levels, stress, obesity, and cigarette smoking.

These findings indicated that it is possible to predict that people who have an ELC are also likely to experience CAD. The detection of an ELC (as an indicator of possible CAD) is a simple, inexpensive, noninvasive screening tool for the detection of CAD.

### Implications

The implications for nursing, with this demonstration of a positive correlation between ELC and CAD are threefold: patient education, nursing practice and future research. This investigation has demonstrated that identification of the individual with the ELC might indicate that that individual may be at risk for CAD. With this information, the nurse would educate the individual toward reducing or eliminating the risk factors for CAD and in so doing, possibly reduce myocardial infarctions. Education of the patient of this correlation would promote increased awareness on the part of the patient, to observe for the ELC in self, siblings, children and/or grandchildren. This self-identification might facilitate earlier self-entrance or entrance of others to the educational

process and reduce delay in treatment, possibly reducing the disease progression.

Nursing practice would be assisted by identification of this positive correlation in that approaches to specific problems might be changed or improved upon. An example of this is the individual with atypical chest pain, that appears to be indigestion, without other adverse symptoms. A positive identification would cause the nurse to immediately consider angina or an MI and take a different, more cautious approach to what appears to be a benign situation. The nurse, in preventive health care practice, is in a position of referral and in such a position is able to refer the client at risk to the physician and/or other supportive disciplines. Hence, increased effectiveness must ensue. The ELC could be used as a screening tool to identify potential individuals at risk in the general population.

### Limitations

Limitations of this study were encountered and suggestions for improvement are fairly numerous. A larger sample size, an increased number of female participants and a larger number of participants in those age groups where the number was below ten would increase reliability. Inclusion of individuals of other than Caucasian, American origin, with different habits, customs and beliefs, to determine whether or not Caucasian American culture increases ELC incidence is recommended. The setting of



the study, in an area with many individuals who follow the Latter Day Saint religious health code, limits the transferability of this study's data as does the male/female ratio.

#### Recommendations for Future Research

The implications for nursing research are numerous and are outlined as follows. Further research that might improve upon, lend credence to, clarify, validate or refute this study include:

1. Observation of individuals of other ethnic origins, e.g., Black, Hispanic, or Oriental, to see whether or not there is an increased prevalence of the ELC in American Caucasians.
2. Evaluation of the occurrence of the right ELC and the left ELC and determination if a correlation exists between the right ELC only and CAD.
3. Observation in a rural hospital to provide a comparison between urban and rural areas.
4. Comparison of men and women for an increased prevalence of the ELC.
5. Further histological studies on the ear-lobe to search for a relationship between the pathological process involved in ELC formation and CAD.
6. Further genetic studies to determine cause of the ELC and CAD.
7. Examination of the influence of the Mormon culture on the incidence of myocardial infarction in women.

8. A longitudinal study of individuals found with the ELC but no history of myocardial infarction. Intervention with education and evaluation of long-term ear-lobe changes and the incidence of MIs.

### Summary

The hypotheses presented for examination were that a positive correlation existed between the presence of a diagonal ear-lobe crease (ELC) and coronary artery disease (CAD) and its risk factors. The rationale for the study was that the demonstration of a positive correlation between the ELC and CAD would provide an indicator of those individuals possibly at high risk for the development of CAD. Recognition of these individuals would do much to facilitate earlier patient education and perhaps reduce or even eliminate one or more of the modifiable risk factors for CAD.

Seventy-seven study subjects who had sustained a myocardial infarction, and 77 control subjects who had not, were randomly selected from a population of hospitalized individuals in an urban hospital. A three-page questionnaire concerning personal and family history of CAD and risk factors for CAD was administered and the ear-lobe observed for presence or absence of a crease.

Corrected chi-square demonstrated strong statistical support for a connection between CAD and the ELC, and age,

hypertension, elevated cholesterol and the ELC. Strength of correlation was supported by Phi and Contingency coefficient measurements.

Recognition of the person at risk for a specific disease entity is an approach that preventive therapy has adopted in its approach to the patient education process, with the inherent intent of educating the individual prior to a morbid episode occurring. The recognition of the individual at risk for CAD would provide the means of prediction, and educational intervention might then reduce that individual's coronary risk.

## APPENDIX A

SUBJECT QUESTIONNAIRE: PERSONAL HISTORY

Diagonal Ear Lobe Crease and Coronary  
Artery Disease

Subject Questionnaire, Identification Number \_\_\_\_\_

Personal History

1. Do you have heart disease? \_\_\_\_\_
2. Do you have coronary artery disease? \_\_\_\_\_
3. Have you ever had a heart attack? \_\_\_\_\_
4. If yes, age first heart attack occurred \_\_\_\_\_
5. Are you overweight? \_\_\_\_\_
6. Do you smoke cigarettes? \_\_\_\_\_
7. Do you have or have you ever had high blood pressure? \_\_\_\_\_
8. If yes, age diagnosed \_\_\_\_\_
9. Do you have or have you ever had elevated blood sugar or diabetes? \_\_\_\_\_
10. If yes, age diagnosed \_\_\_\_\_
11. Do you have or have you ever had elevated cholesterol levels?  
\_\_\_\_\_
12. If yes, age diagnosed \_\_\_\_\_
13. Which of the following do you feel best describes your personality:
  - a. Compulsive, fiercely competitive achievement oriented, involved in multiple activities with deadlines; impatient in slowness in others, sets a rapid work pace. \_\_\_\_\_
  - b. Easy-going, not fiercely competitive not a clock-watcher, not overly impatient, able to relax, does relax. \_\_\_\_\_

## APPENDIX B

SUBJECT QUESTIONNAIRE: FAMILY HISTORY

Diagonal Ear Lobe Crease and Coronary  
Artery Disease

Family History

1. Does (did) your mother have heart disease? \_\_\_\_\_
2. Does (did) she have coronary artery disease? \_\_\_\_\_
3. If alive, age of your mother? \_\_\_\_\_
4. If deceased, did she die from a heart attack? \_\_\_\_\_
5. Age at time of death? \_\_\_\_\_
6. Does (did) your father have heart disease? \_\_\_\_\_
7. Does (did) he have coronary artery disease? \_\_\_\_\_
8. If alive, age of your father? \_\_\_\_\_
9. If deceased, did he die from a heart attack? \_\_\_\_\_
10. Age at time of death? \_\_\_\_\_
11. How many sisters do (did) you have? \_\_\_\_\_
12. Age (s) of your sisters? \_\_\_\_\_
13. Do (did) any of your sisters have heart disease? \_\_\_\_\_
14. Do (did) any of your sisters have coronary artery disease? \_\_\_\_\_  
\_\_\_\_\_
15. If deceased, did any of your sisters die from a heart attack?  
\_\_\_\_\_
16. Age (s) at time of death \_\_\_\_\_
17. How many brothers do (did) you have? \_\_\_\_\_
18. Age (s) of your brothers? \_\_\_\_\_
19. Do (did) any of your brothers have heart disease? \_\_\_\_\_

20. Do (did) any of your brothers have coronary artery disease?

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21. If deceased, did any of your brothers die from a heart attack?

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22. Age(s) at time of death? 

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Thank you.



## APPENDIX C

### SUBJECT INFORMATION SUMMARY

Diagonal Ear Lobe Crease and Coronary  
Artery Disease

Subject Information Summary

1. Identification Number \_\_\_\_\_
2. Age \_\_\_\_\_
3. Sex \_\_\_\_\_
4. Height \_\_\_\_\_
5. Weight \_\_\_\_\_
6. Overweight? \_\_\_\_\_
7. American Caucasian? \_\_\_\_\_
8. Personal history of C.A.D.? \_\_\_\_\_
9. Family history of C.A.D. \_\_\_\_\_
10. Blood sugar elevation or history of diabetes? \_\_\_\_\_
11. If number 10 positive, age diagnosed \_\_\_\_\_
12. Blood pressure elevation or history of hypertension? \_\_\_\_\_
13. If number 12 positive, age diagnosed? \_\_\_\_\_
14. Elevated blood cholesterol or history of elevation? \_\_\_\_\_
15. If number 14 positive, age diagnosed? \_\_\_\_\_
16. Cigarette smoker? \_\_\_\_\_
17. Type A personality? \_\_\_\_\_
18. Ear-lobe crease present? \_\_\_\_\_

Observer Signature \_\_\_\_\_

Date Observed \_\_\_\_\_

## APPENDIX D

### ILLUSTRATION OF ELC



APPENDIX E  
INFORMED CONSENT

Informed Consent

I, \_\_\_\_\_  
           (first name)                    (middle initial)                    (last name)

agree to participate in the Creased Ear-Lobe Study to be conducted by Grace Blodgett, R.N. The purpose of this study is to demonstrate whether or not a positive relationship exists between the presence of Coronary Artery Disease and the presence of a diagonal crease of the ear-lobe. Literature does not fully support this suggestion, is not conclusive and the subject remains debatable.

I understand that:

1. The procedures involve a visual observation of both ear-lobes for the presence of an ear-lobe crease. This observation does not require the ear to be touched.
2. I will complete a personal and family history questionnaire.
3. The investigator will complete a questionnaire on information obtained from the hospital chart and from the completed questionnaire.
4. There are no physical discomforts or risks involved to my person.
5. Any questions I have in reference to the study will be answered by the investigator.
6. An identification number will be given to maintain confidentiality, the key to which will remain locked and be available only to the investigator.
7. Participation is voluntary and may be withdrawn at any time.
8. Holy Cross Hospital, it's physicians and staff are released from any liability associated with this study.

Signature \_\_\_\_\_ Witnessed by \_\_\_\_\_

Date \_\_\_\_\_

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